amounts successively injected as the venoclysis proceeds. The use of antitoxin intravenously introduces a slightly increased risk of treatment, and is applicable to only a small percentage of the patients treated, but it is the only method which will effectively reduce the 5 per cent to 10 per cent immediate mortality which occurs in fulminant cases of meningococcemia. The patient may be given intramuscular injections of adrenal-cortical extracts as a supportive measure.

When treatment has thus been instituted, it is thereafter carried on by means of the sulfonamide alone exactly as heretofore described.

SPINAL PUNCTURE

Spinal punctures are performed principally for the diagnosis of purulent meningitis, and many patients will not require additional spinal puncture for any purpose. Unless the hydrostatics of the subdural space are interferred with by repeated puncture, there will usually be no reason thus to relieve pressure, and subsequent punctures need only occasionally to be performed to confirm clinical improvement or recovery, or to determine the adequacy of spinal fluid sulfonamide levels. A majority of patients proceed so rapidly to evident clinical recovery that confirmatory spinal puncture is unnecessary.

PENICILLIN

Penicillin is demonstrably effective against the meningococcus and has been employed to a limited extent in the treatment of this disease. The response to sulfonamide drug is so satisfactory, however, that at a time when supplies of penicillin are limited, the use of this agent in meningococcus infection should be restricted to the occasional patient who is sensitive to the sulfonamides or the extremely rare instances in which the organism is refractory to sulfonamide treatment. Penicillin may be employed in dosage of 10-20,000 units, given intramuscularly every three hours. It is improbable, although apparently not definitely demonstrated, that the introduction of penicillin intrathecally is necessary or desirable as a routine of treatment of meningococcus disease. Unquestionably penicillin, in common with almost every other agent which may be introduced intraspinally, produces meningeal irritation and spinal fluid pleocytosis, which is confusing to the clinician and is therapeutically disadvantageous.

There is little doubt that at the present time the sulfonamide drugs are an almost completely adequate agent for the control of meningococcus disease.

PROPHYLAXIS

The communicability of endemic meningococcus disease is extremely low and contact cases are the exception. When there has been intimate contact within a family it is sometimes appropriate to safeguard contacts by the administration of sulfadiazine in doses of about one-half grain per pound per 24 hours for two or three days, which acts as an effective prophylactic. In the epidemic form of the disease in which bacteriological studies have revealed a high carrier rate in army camps or barracks, the use of similar dosage of sulfadiazine for 48 to 72 hours in the entire personnel will reduce the number and viability of organisms which are harbored, and this has been shown to be an effective measure in the arrest of epidemics.

384 Post Street.

No education, no refinements of civilization can compensate a people for the loss of their hardy virtues. The greatest danger of a luxurious civilization is that it is likely to lead a people to lose their fighting edge.—Theodore Roosevelt.

ACUTE ANTERIOR POLIOMYELITIS*

HENRY BRAINERD, M. D.
San Francisco

MUCH attention has been focused on the problems of poliomyelitis by the controversy aroused following the introduction of the Kenny concept,[1] and, more recently, by the epidemic prevalence of the disease.

The etiological agent is a filterable virus which has been isolated from the nasopharyngeal secretions[2] and stools[3] of affected patients, as well as from the excreta of apparently healthy individuals. The virus may occur in sewage,[4] probably in contaminated milk, food and water, and may be passively carried by the housefly.[5]

The concept of entry through the olfactory end-plates has largely been discarded. Portals of entry may presumably be either through the gastroenteric tract[6] or the nasopharyngeal mucosa,[7] whence the virus is transmitted along autonomic nerve trunks to the central nervous system.

Dissemination of the virus within the central nervous system is widespread, but certain sites of predilection are most prominently involved. Many cells are damaged temporarily by the virus and later recover, but some are permanently destroyed in a spotty fashion. In those cases exhibiting flaccid paralysis, anterior horn cell destruction is marked. In numerous cases in which muscle spasm predominates lesions of the small internuncial neurones of the anterior horn have recently been described.[8]

CLINICAL OBSERVATIONS

In the light of recent observations, both the Kenny concept and the older orthodox concept appear to suffer from over-simplification. The relationship of the pathological physiology of the disease to the clinical picture is now in a state of revision.

Following exposure of a group of individuals to poliomyelitis several alternative courses may ensue. In most cases no infection will occur due to the presence of neutralizing antibodies. A smaller group of exposed individuals, after an incubation period of, most commonly, a week, but varying from four to eighteen days, will develop a systemic disease of no specific diagnostic features. Moderate fever, accompanied by nausea and vomiting, diarrhea or constipation, abdominal pain, or symptoms of infection of the upper respiratory tract, may occur. These symptoms, persisting for a few days, may constitute the entire illness, and the diagnosis may be only suspected clinically, unless the virus be isolated or a rise in neutralizing antibodies be demonstrated.

In a still smaller group of exposed persons the disease may progress to a point where it becomes clinically recognizable as involving the central nervous system. Evidences of nervous involvement may develop during the course of the prodromal illness described above, or may succeed it after an asymptomatic interval of several days. Rarely paralysis may occur abruptly without a disease of onset.

In a majority of persons in whom nervous system invasion is manifest, muscle spasm dominates the picture.[9] Although the pathogenesis of this phenomenon is the subject of debate,[8,10,11,12] clinically it is quite striking, and may of itself be the cause of persistent disability. Spasm consists of hyperreactivity of muscles to stretch stimulation, and is manifested by limitation of motion of

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* One of several papers in a symposium on "Communicable Diseases." Papers in the symposium have been collected by Dr. Edward B. Shaw, San Francisco.

From the Divisions of Medicine and Pediatrics, University of California Medical School; Department of Pediatrics, Stanford University Medical School; and Department of Public Health, City and County of San Francisco.

the part due to pain and contraction of the muscle. In every instance spasm of back muscles is present[9] and is characterized by rigidity of the spine and resistance to flexion. Patients commonly assume the "tripod" posture on sitting up. Spasm of the posterior cervical and hamstring muscles is almost as common. This stage or degree of involvement is commonly termed the "preparalytic stage," and the symptoms have usually been ascribed to meningeal irritation. However, it must be emphasized that not only does paralysis frequently not follow these manifestations, but also the signs may persist for months after all possibility of meningeal irritation is passed, and may indeed occur in the absence of spinal fluid pleocytosis.[9] Spasm may exist in any other muscles, although less commonly, including particularly the calf muscles, pectorals and muscles of the upper arm. The associated symptoms under these circumstances are: fever, usually of a moderate degree, although occasionally absent or very high, headache, nausea and vomiting, pains in the back and neck, and pains and sometimes paresthesias in the extremities. The patient is frequently drowsy when undisturbed, but irritable when examined.

In a smaller number of cases, appreciable muscle weakness is present in addition to spasm. Much debate exists at present over the cause of the muscle weakness seen in poliomyelitis.[8,10,11,12] Certainly at least some is due to anterior horn cell damage or destruction. In such cases the involvement may vary in type, location and degree. True flaccid paralysis of lower motor neurone type associated with atrophy, fibrillary twitching, and reaction of degeneration, may involve one muscle, or one or more extremities. In addition, frequently a weak muscle may exhibit a striking degree of spasm. Sister Kenny describes the condition of "mental alienation,"[1] wherein a muscle opposed to a muscle in spasm becomes weak or functionless. This she ascribes to absence of nervous impulses to the muscle without, however, any lesions of the nervous pathway. Whether this phenomenon alone can produce, if untreated, permanent weakness of a muscle, is open to question. On the other hand, clinically such a situation does seem to contribute to muscle weakness, but would appear to be due simply to the braking action of the opposing muscle and inhibition due to pain on stretching a muscle in spasm. It seems likely that weakness and flaccidity of a muscle cannot exist without at least temporary interference with its motor innervation on an organic basis, but that mental alienation may be coëxistent or superadded, and produce permanent disability if allowed to go untreated.

In addition to spasm and loss of function of muscles, incoördination of muscle action is often a cause of much disability. An involved muscle may contract[9,11] only in part of its belly, or from its origin, or most striking, the opponent of the muscle may contract. Frequently strong muscles are substituted for weak, producing a dysrhythmic motion.

The tendon reflexes, contrary to commonly-accepted precepts, are often normal or hyperactive when spasm is present in the effector muscle of the reflex arc.[9] Depressed reflexes are seen only in association with marked muscle weakness.

Involvement of the medulla usually occurs in combination with back and neck muscle spasm, or with more extensive cord involvement. Disturbance of the respiratory center is manifested by respirations which are irregular in depth and rhythm, although the patient is able to expand his chest fully, in contrast to respiratory difficulty due to spasm or weakness of the muscles of respiration. Paralysis of swallowing and phonation may be observed. Facial nerve lesions may be either peripheral or central in type. Deviation of the tongue may occur. Lesions of other cranial nerves are uncommon. Marked changes in sensorium are seen occasionally when the disease involves higher centers, and rarely ataxia and athetosis may signify cerebellar or basal ganglion involvement. Coma and convulsions are exceedingly rare.

Thus the clinical picture may consist of a wide variety of types and severity of muscle involvement. Mild spasm, involving the back and neck muscles, is frequently the entire extent of the disease. On the other hand any group of combinations of spasm and weakness or alienation may be present. Generally the maximum involvement occurs soon after the onset, but spasm or weakness may progress for as long as a week, especially if fever is present.

Poliomyelitis must be differentiated from: 1) other infections of the central nervous system, such as meningitis and encephalitis; 2) diseases producing muscle spasm or limitation of motion of parts, such as rheumatic fever and arthritis; and 3) from diseases producing a lower motor neurone type of muscle weakness, such as peripheral neuritis.

LABORATORY FINDINGS

Although a mild degree of leukocytosis is commonly observed, the examination of the cerebrospinal fluid is the only simple laboratory aid in the diagnosis. In most cases an increase in cells is noted. Early, polymorphonuclear leukocytes may predominate, but later lymphocytes are more numerous. The total cell count is generally less than 300 per cu. m.m., but occasionally as high as 1,000 cells or more may be present. An associated increase in globulin in about half the cases is the only other finding of note. The pressure is rarely greatly elevated. The sugar and chloride content of the fluid is characteristically normal, and this may be of aid in the differentiation from bacterial meningitis. In a significant number of clinically typical cases the spinal fluid may be normal on repeated examinations, and the diagnosis may only be made by continued observation of the patient.[9,10] Isolation of the virus from the nasopharyngeal washings or stools is not a practical procedure under ordinary circumstances. A rise in protective antibodies during the interval from onset to convalescence may allow a retrospective diagnosis in atypical cases.

TREATMENT

The Kenny treatment[1] appears to be the method of choice. The aims of the treatment are to eliminate muscle spasm, reeducate muscles whose function is disturbed, and to eliminate incoördinated use of muscles. Regardless of the origin of the muscle dysfunction observed in poliomyelitis, this treatment is entirely rational and, moreover, successful.

The patient is placed in a neutral supine position on a bed under whose mattress is a plywood board. A vertical footboard is inserted several inches beyond the foot of the mattress. Packs consisting of woolen material, cut to shape, and wrung twice out of boiling water are applied to all muscles in spasm. These are then covered with waterproofing and finally blanketing. Care is taken not to interfere with movement of joints. The foments are applied six times daily at the outset, but may later be reduced to four. The extreme degree of moist heat (approximately 170 F.) followed by rapid cooling has been found the most efficient means of relaxing spasm. Packs are continued until all muscle spasm has disappeared. When calf muscle spasm is alleviated, the feet are placed against the foot board to stimulate proprioceptive reflexes.

From the beginning, once or twice daily, the joints are put through whatever range of passive movement which can be encompassed without pain. As spasm subsides, reëducation of muscles begins. The patient is asked to

concentrate on the insertion and movement of the muscle being reëducated and then the movement of the muscle is simulated passively. Later after several passive movements, the patient is allowed to attempt active contraction of the muscle. Great care is taken to prevent incoördination. This program is continued once or twice daily as long as required, since improvement may occur over a period of years. A thorough knowledge of muscle anatomy and function is necessary on the part of the physical therapeutist.

In patients exhibiting respiratory difficulty due to weakness or spasm of the muscles of respiration, frequent packing is the treatment of choice, but the respirator is occasionally necessary. If the respiratory center is involved sufficiently to cause hypoxia, the respirator is of definite, although limited value. Patients with pharyngeal paralysis do better with very frequent small feedings, but occasionally tube feeding is necessary, supplemented by parenteral fluids. Aspiration of secretions in the phraynx must be done frequently. Sulfonamides are of possible value in the prevention of pneumonia. Convalescent serum is of doubtful value. Prostigmine appears to relax the muscle spasm of poliomyelitis, but its exact place in the treatment is not clear.[9]

The prognosis in poliomyelitis is dependent on many factors. Cases exhibiting muscle spasm without appreciable weakness uniformly do well with the Kenny treatment, although they would probably recover, although more slowly, without it. Patients exhibiting muscle weakness may all be expected to recover function, at least in part, and without significant deformity. Bulbar cases have a 50 per cent fatality rate, almost always due to respiratory failure or its complications, but those who recover usually do so relatively rapidly and completely.

U. C. Medical Center, Third and Parnassus.

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Medical statesmanship comprises the art of changing the health condition of a community, county, or state from what it is to what it ought to be.—Walter F. Donaldson.

Life is short and art is long.
—Hippocrates, *Aphorisms.* No. 1. Hippocrates is speaking of the art of healing.

TROPICAL DISEASES*

HERBERT G. JOHNSTONE
San Francisco

THE present world-wide conflict has suddenly revealed to the medical profession of the United States that tropical diseases no longer should be regarded a matter of text book consideration. The American troops are now serving in many foreign parts, with the majority in the tropics and in regions bordering on the tropics. The ever-increasing number of military and navy personnel returning to our shores with tropical infections, infrequently or rarely encountered in most of the sections of our country, presents a distinct challenge not only to the medical departments of the Army and Navy, but to the civilian practitioner.

It is apparent that this problem will not be limited to the duration of the war, but its many ramifications will, no doubt, cause increasing concern in years to come. The present trend of military operations indicates that, in all probability, certain detachments of American troops will be retained in the tropics for some time following the close of the war. Furthermore, it is not unlikely that many civilians representing governmental, industrial and educational agencies will carry on their activities in tropical and subtropical countries.

Following the return of the first groups of Army and Navy personnel sent home for the treatment of parasitic infections acquired in tropical combat zones, alarms were sounded warning that these men would serve as foci of widespread epidemics of catastrophic proportions. Speculations were rife as to what disastrous consequences would be produced by masses of disease-harboring troops dispersed throughout the nation. Time will determine whether or not we are justified in becoming unduly alarmed about these prospects. Natural barriers in addition to those created by our various public health agencies will continue to function just as they have in the past. The effectiveness of created barriers, however, is lessened due to the relaxed supervision existing under present war conditions.

Although there is little justification for the almost hysterical approach to the problem confronting us, it should be emphasized that there is inherent danger in the uncontrolled widespread dispersion, during the war and postwar period, of persons with active or latent tropical disease. Instances may be cited to show that new endemic foci can be established following the entrance of infected individuals into a community in which the disease in question had not previously existed. The introduction of malignant tertian malaria into Connecticut by members of the National Guard who had acquired infections in Southern camps during the Spanish-American War and the Aurora, Ohio, epidemic of 1934, which followed the importation of a single case of malaria, are examples worthy of notice. Of the several millions of men now enlisted in the armed forces the greater proportion are engaged in areas noted for the prevalence of some of the most terrible scourges of man: Africa, the Mediterranean littoral, the Middle East, India, China, and the islands of the Southwest Pacific. Our military operations in previous wars, with the exception of the Philippine campaign during the Spanish-American War, have been limited to the temperate regions, and the troops engaged were not subjected to the disease hazards which are ever

* One of several papers in a symposium on "Communicable Diseases." Papers in the symposium have been collected by Dr. Edward B. Shaw, San Francisco.

From the Department of Bacteriology, University of California Medical Schools, San Francisco.